No increased mortality in later life for cohorts born during famine

Väinö Kannisto,[1] Kaare Christensen,[1,2] James W. Vaupel[1,3]

[1] Odense University Medical School, Winslowparken 17, DK-5000 Odense C, Denmark

[2] The Danish Epidemiology Science Centre, The Steno Institute of Public Health, Department of Epidemiology and Social Medicine, Aarhus University Hospital, Aarhus, Denmark.

[3] Sanford Institute, Duke University, Durham, NC.

Reprint requests to: Kaare Christensen

Odense University Medical School

Winslowparken 17

DK-5000 Odense C

Denmark

Abstract

Nutrition early in life may influence adult mortality. In particular, the fetal-origins hypothesis suggests that an individual's nourishment before birth and during infancy programs the development of risk factors for several important diseases of middle and old age. The present study was designed to evaluate the impact of extreme nutritional deprivation in utero, infancy, and early childhood on mortality in later life. We analyzed the survival of the cohorts born in Finland during the severe 1866-68 famine and during five immediately preceding and five succeeding years. A total of 331,932 individuals born prior to the famine, 161,744 individuals born during the famine, and 323,321 individuals born after the famine were included. We assessed survival by cohorts from birth to age 17 and from age 17 to 40, 60 and 80, as well as average length of life after age 80. Survival from birth to age 17 was significantly lower in cohorts born before and during the famine than in those born after it (Males: 0.566 vs 0.671, a difference of 0.105 (95% CI 0.102-0.108); females 0.593 vs 0.692, a difference of 0.099 (95% CI 0.096-0.102)). At subsequent ages, including old age, mortality was practically identical in the famine-born cohorts and in those five cohorts born before and after the crisis. For both males and females, survival from 17 to 80 and mean remaining lifetime at age 80 were very similar across the 13 cohorts studied. These findings suggest that cohorts subject to prolonged and extreme nutritional deprivation in utero, infancy, and early childhood suffered an immediate rise in mortality but after the crisis had passed, carried no after-effects that affected their survival at any time in later life.

Keywords: Fetal-origins hypothesis, mortality, famine, cohort effects.

The lingering effects of early-life conditions on adult mortality have long intrigued epidemiologists and demographers. Some 60 years ago Kermack et al.(1) analyzed death rates in Great Britain and Sweden and concluded that cohorts carry their later-life mortality with them from childhood. Some other studies of 19th and 20th century populations have shown similar results, but it is very difficult to disentangle the effect of belonging to a cohort and the effect of changing living conditions with time (period effects) when both cohorts and periods have shown substantial gradual mortality improvement (2).

Nutrition and infections have been the most prominent among hypothesized early-life conditions of importance for adult mortality. Due to lack of data on individuals' early-life nutrition and infections as well as later life mortality, adult height has often been used as a proxy for these early-life exposures (2-4), although it is likely that genetic factors make a very significant contribution to the variation in height (5).

In a long series of papers D.J.P. Baker and co-workers have reported on a follow-up of the individuals found in a large British source of detailed birth and infant records from the beginning of the century (6-9). These studies have indicated that low growth rates in utero and during infancy are associated with adverse health outcomes at adult ages. In particular, a strong relationship between reduced growth early in life and high death rates for cardiovascular diseases has been reported (6-9). Based on these observations D.J.P. Baker and co-workers have proposed "the fetal-origins hypothesis", which asserts that a baby's nourishment before birth and during infancy program its susceptibility to diseases late in life. Positive associations between low growth rate in

utero and during infancy - measured as weight at birth and 1 year - have been reported not only with cardiovascular diseases, but also with diabetes mellitus, hypertension, raised serum cholesterol, abnormal blood clotting, autoimmune diseases and even suicide (6-10). Because these associations involve the causes of most adult deaths, nutrition in utero and infancy may be crucial to adult health and mortality.

The fetal-origins hypothesis suggests optimism concerning future population levels of diseases since an increase in birth weight has been observed in a number of countries during the last decades and interventions to increase birth weight seem feasible (11). Possibilities for testing the fetal origins of adult diseases are, however, limited at present as information on both nutritional intake (or even birth weight) and disease incidence over the entire adult life is lacking (12). Most studies have replaced disease incidence by disease prevalence or mortality, and follow-up has usually been limited to certain age ranges. The lack of birth weight data has been addressed by studying twins, who experience considerable growth retardation during the third trimester (13,14).

Studies of cohorts that have experienced famine in utero and during infancy can shed light on the influence of fetal programming on adult diseases and the more general hypothesis that early-life conditions affect late-life mortality. From the extensively analyzed Dutch famine of 1944-45 it is known that third trimester exposure to famine reduces fetal growth including birth weight (15). One of the greatest well-documented mortality crises in population history is the 1866-68 famine in Finland. Three successive crop failures, the last two total losses in many areas, caused widespread famine followed by epidemic diseases. As a result, mortality increased sharply at all ages. The excess

mortality during the three years carried away no less than 8% of the total population of the country; infant mortality rose to 40% in 1868. With a good crop in the autumn of 1868 the situation returned to normal late in 1868 and the following years (16).

The 1866-68 Finnish famine thus represents a well defined time period of extremely serious and prolonged malnutrition among a population including expectant and lactating mothers, infants, and young children. The official statistics of Finland contain comprehensive, accurate information on the catastrophe and its aftermath all the way to the extinction of the affected generation. Based on the fetal-origins hypothesis and the line of epidemiological research that emphasises the cohort effects of early-life conditions on adult mortality, it can be expected that the cohorts born during or just before famine would experience increased mortality in later life. In the present study the survival of the three Finnish famine cohorts from 1866-68 is compared with the five immediately preceding cohorts, which were also affected by the famine, but at ages 1 to 7, as well as the five succeeding cohorts, which were born after the famine.

Materials and methods

Our study is based on Finnish vital statistics, which include information on annual live births and deaths. Although comprehensive vital statistics for Finland go back to 1751, two factors complicate measurement of survival of the 1861-73 cohorts. First, deaths have been cross-classified on an individual level by year of birth and year of age only since 1878. Therefore, the youngest age for which the number of survivors can be determined for the 1861-73 cohorts is age 17. Second, migration statistics were unsatisfactory as they included passport takers whether they actually emigrated or not, and took no account of returners. From about 1890 to 1914 large-scale emigration to North America took place (17).

To overcome these limitations in the data source, the following five survival indicators for males and females were chosen: Survival from birth to age 17 and then to ages 40, 60 and 80 and average length of life after age 80 years.

Survival from birth to age 17: The annual number of live births is directly available from the vital statistics. As the births were given for both sexes combined, the officially recorded sex ratio (1.048) for 1861-70 was used to estimate the size of the two sex-groups. The number of survivors at age 17 was obtained from death counts and the census population at the end of 1880: To the census population of the 1861-63 cohorts the deaths after exact age 17 but before the census were added, while for the 1864-73 cohorts the deaths which took place after the census but before age 17 were subtracted. This procedure converts the census age to age 17. Published estimates of age-specific death rates for the period are consistent with our estimates (18).

Survival from age 17 to age 80. From age 17 on, the mortality history of the cohorts under study, excluding emigrants, is accurately known because the age of every deceased person was routinely verified against the population register which, in turn, was based on birth registration. Net emigration in each cohort after age 17 was calculated as the difference between the number of survivors at age 17 and the number of all subsequent deaths recorded for the cohort. The latter figure represents non-migrants at 17, and their subsequent mortality at any given age is readily obtained from the number of deaths.

Average length of life after age 80. After age 50 and even more after 80 international migration was negligible and the cohorts were gradually reduced only by death. The cohorts born in 1861-73 are now extinct as the last survivor, a woman born in 1872, died in 1978. Survival after age 80 is expressed as the average number of years actually lived.

Results

Figure 1 illustrates the sharp increase in mortality during the famine and the less dramatic drop in the crude birth rate. Table 1 shows the follow-up of cohorts born before, during and after the 1866-68 famine. Survival from birth to age 17 (Figure 2) was significantly lower in cohorts born before and during the famine than in those born after it (males: 0.566 vs 0.671, a difference of 0.105 (95% CI 0.102-0.108); females 0.593 vs 0.692, a difference of 0.099 (95% CI 0.096-0.102)). The parallel age-specific mortality curves given in Figure 3, which are based on published estimates (18), indicate that no pronounced cohort effects were present before age 17. The curves rise and fall together in successive calender years; there is no apparent indication that high mortality in a younger age category is followed by high mortality in older age categories as cohorts grow older.

Table 2 summarizes the indicators of adult survival for the three famine cohorts and the three and five cohorts immediately preceding and following the famine cohorts. The indicators of adult survival were all approximately equal across these three groupings of cohorts, rarely differing by more than 1%. Some survival indicators were *higher* for famine-born cohorts than for the controls. The remarkable stability of the survival indicators across the different cohorts is also shown in Table 1 and Figure 4. It is clear that the cohorts born before or during the famine do not differ substantially from the control cohorts born after the famine in any of the adult survival indicators. The survival curves for the famine cohorts and the control cohorts were virtually identical after childhood.

Net emigration after age 17 was 9.5% for males in the famine cohorts compared

with 10.0% for the ten cohorts born before and after the famine; the corresponding numbers for females were 4.9% vs 5.6%. These small differences are statistically significant ($p < 0.01$) because the population studied is so large.

Discussion

Our study provides information on the long-term health consequences for cohorts born during or just before a famine. The fetal-origins hypothesis asserts that an individual's nourishment before birth and during infancy program the development of risk factors for a number of specific diseases in adulthood (in particular cardiovascular diseases). Our data are on a cohort basis and information on individual birth weight is not available. A nation-wide famine, however, that carried away no less than 8% of the total population in a non-industrialized country is likely to affect the nutrition of the majority, including most of the pregnant women and their babies. A limitation in our study was the inability to assess cause-specific mortality due to the historical nature of the cohort investigated. Data on cause-specific mortality from Nordic countries (19) are available back to the 1930s, and even at that time cardiovascular diseases were among the most frequent causes of death. Substantially increased cardiovascular mortality should be detectable in a sample of 160,000 cases born during the famine and 650,000 individuals born in the preceding and following five year periods. Furthermore, the growing number of other reported adverse health outcomes linked to reduced growth early in life would also be expected to increase the mortality of the famine cohorts.

A limitation in the demographic data is that survival from birth to age 17 may be underestimated due to emigration. As the emigration to North America mainly took place from about 1890 to 1914 (17) it is unlikely to have had a substantial effect before age 17 on cohorts born between 1861 and 1873. Approximately 10% of the males and 5% of the females were lost to follow up during the lifeterms of both the famine cohorts and the

adjacent cohorts due to emigration. There were slightly more emigrants in the famine cohorts. Because the very weakest in the population probably did not emigrate, this small difference might tend to decrease survival for the non-emigrated famine cohorts. Considering the small size of the emigration group in the different cohorts, extremely large differences in survival for the emigrants in the famine cohorts and the adjacent cohorts are needed to affect the overall result, which shows practically identical survival indicators in adulthood. Therefore, emigration is unlikely to have any significant impact on the survival indicator comparisons.

A remarkable similarity in the survival indicators throughout adult life was observed for the famine cohorts and the cohorts born both before and after the famine. Survival after ages 60 and 80 shows a slight tendency to increase due to a secular trend of mortality decline but the famine-born cohorts fail to stand out from the general trend.

There is a potential for selection biases in the present study, because survival from the famine is likely to be related to factors such as fitness, social class and education which are possible predictive of longevity. In Finland of the 1860s there existed a small, relatively affluent and well-educated class, mainly in the cities. This was, however of small importance because Finland was then 93 percent rural. A great majority of the rural population was engaged in farming, animal husbandry and other occupations linked to farm life. A factor that counteracted normal selection was that the excess mortality during the famine was heaviest in the more prosperous agricultural areas South and West from the geographical center of the country and considerably smaller in the poorer Northern and Eastern regions, as well as on the relatively urbanized South coast (in the late 19th

century mortality was higher in urban than rural areas). Furthermore, in the pre-famine cohorts about 40 percent of girls and 43 percent of boys died before age 17. When this percentage rose in the worst-hit cohorts to 44-47 percent, it did not give much room for additional selection to operate. Any study of the lingering effects of adverse conditions early in life is confronted with this kind of selection problem. Analyses of the effect of fetal and infant conditions on adult diseases and mortality inevitably excludes cases who die before adulthood.

In a recent book, D.J.P. Barker (20) discusses the importance of environmental factors after infancy in relation to the fetal-origins hypothesis. Poor nutrition in fetal and early infant life is hypothesised to be detrimental to the development and function of the β-cells of the islets of Langerhans and to change muscle tissue leading to insulin resistance. It is further suggested that as long as an individual undernourished in utero and infancy remains undernourished throughout adult life, the glucose-insulin metabolism is adequate, whereas a sudden move to good or excess nutrition will trigger diabetes. It is argued that both nourishment in utero and infancy as well as adult risk factors play a role in the etiology of cardiovascular diseases, but "it is not known how important adult overnutrition is in determining coronary heart disease compared with fetal undernutrition". The complete lack of long-term effects of the famine in the Finnish 1866-68 cohorts suggests that if the fetal-origins hypothesis is true, environmental factors which emerged during the 20th century are necessary to trigger the effect of fetal and infant undernourishment on programming of adult diseases.

The Dutch famine cohorts 1944-45 provide an additional data source on the long

term consequences of fetal and infant nourishment (15). In a historical follow-up study of 300,000 19-year-old men exposed to the famine of 1944-45 it was found that famine experienced during the last trimester of pregnancy and the first months of life produced significantly lower obesity rates, whereas during the first half of pregnancy exposure resulted in significantly higher obesity rates (21). These famine cohorts are still rather young for investigation of cardiovascular disease incidence, but future studies will provide powerful tests of the fetal-origins hypothesis in a 20th-century cohort.

Twin studies have shown that birth weight *per se* is not linked to adult mortality (13,14) but birth weight, and in particular twin birth weight, may be a poor proxy for measurement of nourishment in utero and infancy. However, when no effect of prolonged and extreme nutritional deprivation of mothers and babies can be detected in a large scale follow-up of mortality, it seems unlikely that a baby's nourishment before birth and during infancy *per se* is crucial to adult health. The associations found between birth weight and adult morbidity and mortality in the British (6-10,20) and Norwegian studies (22) could have other explanations than a causal link. An obvious possibility is that factors linked to social, family, and environmental conditions are behind both low birth weight and a number of adult diseases, such as cardiovascular diseases. Controlling for social class in studies of the fetal-origins hypothesis may not be sufficient, because income and living standards vary widely within social class groups (23). Another possibility is that some genes have pleiotrophic effects which increase the risk for both low birth weight and diseases later in life.

The present study suggests that cohorts subject to prolonged and extreme

nutritional deprivation in utero, infancy, and early childhood suffered an immediate rise in mortality but after the crisis had passed carried no after-effects that reduced their survival in adult life.

Acknowledgements

This work was supported by grant no. PO1-AG08761 from the U.S. National Institute on Aging. We are grateful to Jørn Olsen for helpful suggestions, and to Kirill Andreev for his help in preparing this manuscript.

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TABLE 1. FOLLOW-UP OF COHORTS BORN BEFORE. DURING AND AFTER THE 1866-68 FAMINE IN FINLAND

	COHORT												
	1861	**1862**	**1863**	**1864**	**1865**	**1866**	**1867**	**1868**	**1869**	**1870**	**1871**	**1872**	**1873**
MALE													
Live births	34 045	33 940	33 252	36 488	32 125	30 115	30 274	22 390	29 881	32 620	34 062	33 869	35 012
Deaths. age 0-16	14 572	15 423	14 097	15 252	14 242	13 362	14 249	8 437	8 945	10 007	10 971	11 951	12 597
Survival ratio 0-17	0.572	0.546	0.576	0.582	0.557	0.556	0.529	0.623	0.701	0.693	0.678	0.647	0.640
Reached age 17	19 473	18 517	19 145	21 236	17 883	16 753	16 025	13 953	20 936	22 613	23 091	21 918	22 415
Net emigrants after age 17	1.879	1.668	1.614	1.981	1.668	1.703	1.503	1.254	2.160	2.230	3.038	2.119	2.363
percent	9.6	9.0	8.4	9.3	9.3	10.2	9.4	9.0	10.3	9.9	13.2	9.7	10.5
Non-migrants at 17	17 594	16 849	17 531	19 255	16 215	15 050	14 522	12 699	18 776	20 383	20 053	19 799	20 052
40	14 621	13 879	14 564	15 972	13 576	12 520	11 983	10 464	15 574	16 999	16 664	16 413	16 693
60	9 970	9 527	9 871	10 795	9 233	8 550	8 227	7 066	10 572	11 446	11 206	11 149	11 205
80	2 012	1 948	1 948	2 079	1 887	1 687	1 693	1 525	2 316	2 558	2 403	2 407	2 415
Survival ratio *													
17-40	0.831	0.824	0.831	0.829	0.837	0.832	0.825	0.824	0.829	0.834	0.831	0.829	0.832
40-60	0.682	0.686	0.678	0.676	0.680	0.683	0.687	0.675	0.679	0.673	0.672	0.679	0.671
60-80	0.202	0.204	0.197	0.193	0.204	0.197	0.206	0.216	0.219	0.223	0.214	0.216	0.216
Mean lifetime after													
17	43.10	42.84	42.82	42.70	43.07	42.86	42.69	42.57	42.77	42.86	42.68	42.72	42.66
80	4.68	4.63	4.78	4.88	4.84	4.78	4.97	4.98	4.83	4.96	4.82	4.93	4.90

	COHORT												
	1861	**1862**	**1863**	**1864**	**1865**	**1866**	**1867**	**1868**	**1869**	**1870**	**1871**	**1872**	**1873**
FEMALE													
Live births	32 489	32 388	31 731	34 819	30 655	28 738	28 890	21 367	28 514	31 128	32 505	32 320	33 410
Deaths. age 0-16	13 008	13 491	12 551	13 635	12 902	12 254	12 652	7 648	7 890	8 826	9 907	10 611	11 430
Survival ratio 0-17	0.600	0.583	0.604	0.608	0.580	0.574	0.562	0.642	0.723	0.716	0.695	0.672	0.658
Reached age 17	19 481	18 897	19 180	21 184	17 753	16 484	16 238	13 719	20 624	22 302	22 598	21 709	21 980
Net emigrants after age 17	1.167	1.164	894	958	687	812	893	573	1.337	1.202	1.452	1.338	1.436
percent	6.0	6.2	4.7	4.5	3.9	4.9	5.5	4.2	6.5	5.4	6.4	6.2	6.5
Non-migrants at 17	18 314	17 733	18 286	20 226	17 066	15 672	15 345	13 146	19 287	21 100	21 146	20 371	20 544
40	15 424	14 894	15 319	16 854	14 431	13 167	12 867	11 029	16 147	17 823	17 860	17 195	17 327
60	12 186	11 759	12 089	13 301	11 385	10 466	10 275	8 686	12 797	14 208	14 148	13 735	13 787
80	3 774	3 661	3 893	4 260	3 704	3 396	3 431	2 859	4 338	4 854	4 745	4 762	4 781
Survival ratio *													
17-40	0.842	0.840	0.838	0.833	0.846	0.840	0.839	0.839	0.837	0.845	0.845	0.844	0.843
40-60	0.790	0.790	0.789	0.789	0.789	0.795	0.799	0.788	0.793	0.797	0.792	0.799	0.796
60-80	0.310	0.311	0.322	0.320	0.325	0.324	0.334	0.329	0.339	0.342	0.335	0.347	0.347
Mean lifetime after													
17	47.03	46.89	46.90	46.68	47.13	47.11	47.11	46.97	47.02	47.45	47.31	47.52	47.51
80	5.25	5.30	5.39	5.39	5.33	5.36	5.33	5.37	5.42	5.35	5.31	5.47	5.53

* For all survival ratios the SE is in the interval 0.003 to 0.005

TABLE 2. Comparison of adult survival indicators in famine and control cohorts.

Sex and survival indicator	3 famine cohorts	6 * control cohorts	10 * control cohorts
MALE			
Survival ratio **			
17-40	0.827	0.832	0.831
40-60	0.682	0.676	0.678
60-80	0.206	0.208	0.209
Mean lifetime after			
17	42.71	42.82	42.82
80	4.91	4.85	4.82
FEMALE			
Survival ratio **			
17-40	0.840	0.841	0.841
40-60	0.794	0.792	0.792
60-80	0.329	0.330	0.330
Mean lifetime after			
17	47.06	47.08	47.14
80	5.35	5.36	5.37

* The control cohorts consist of the 3 (5) immediately preceding and following birth cohorts.

** For all survival ratios the SE is in the interval 0.001 to 0.003

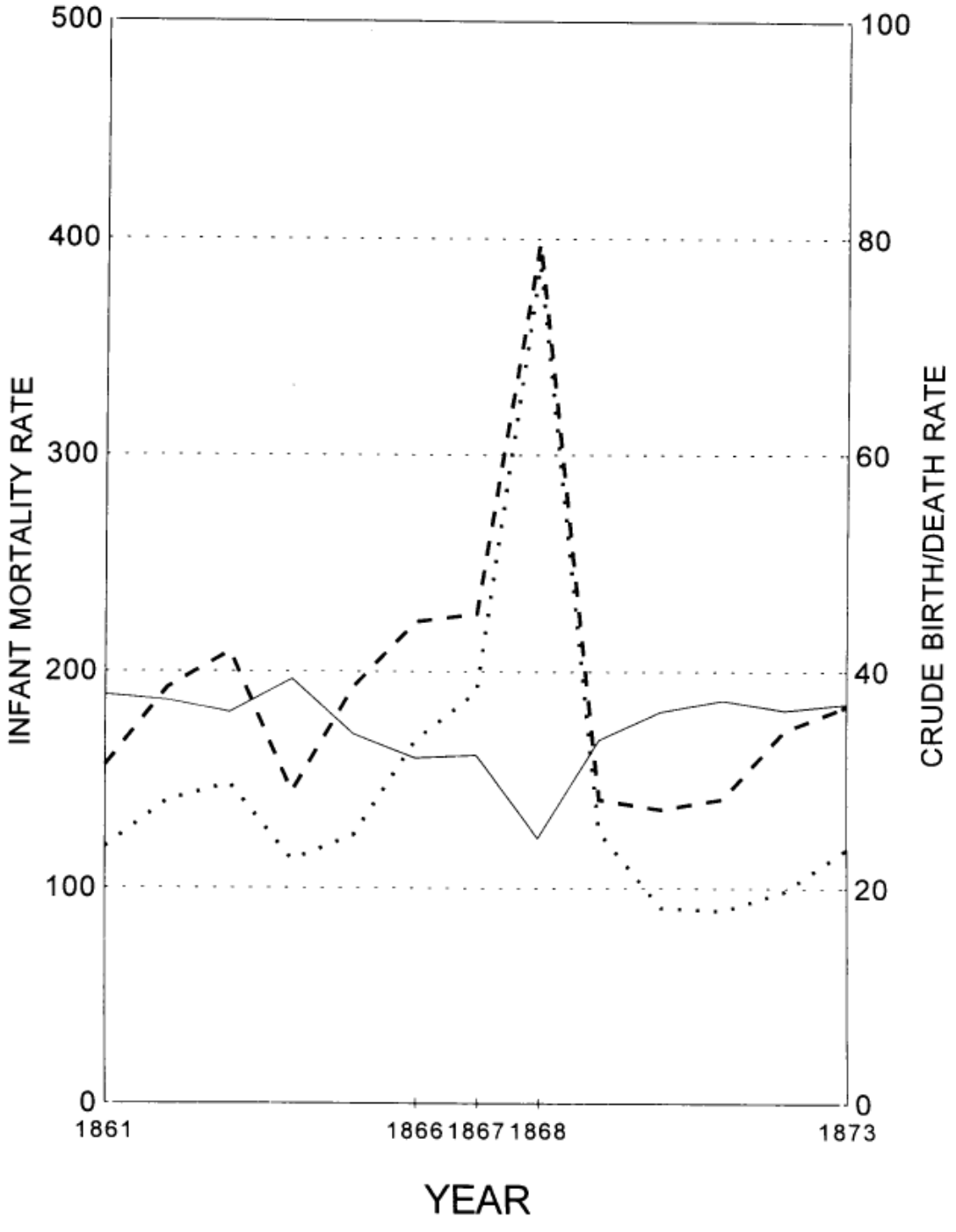
Legends

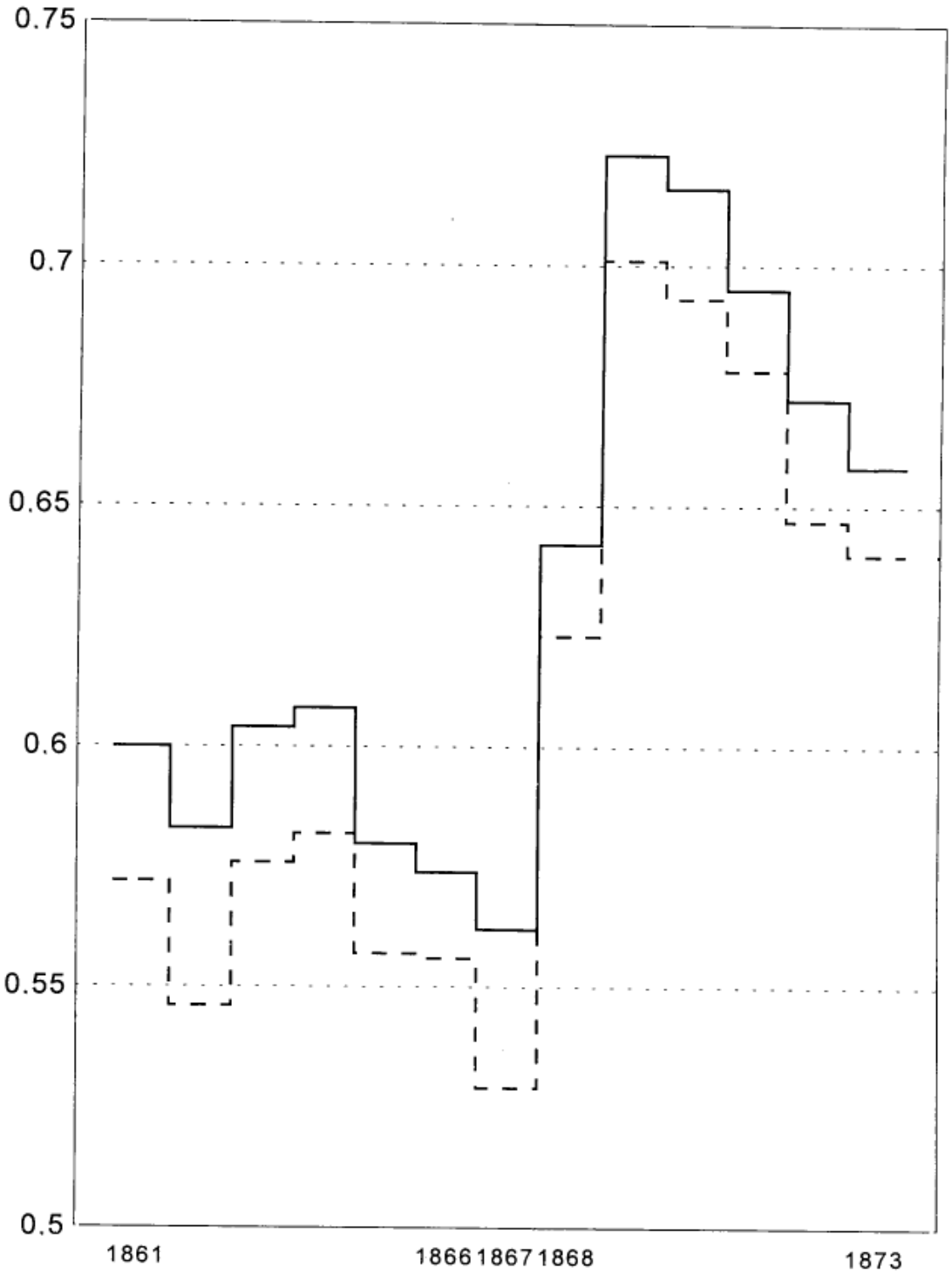
Figure 1. Infant mortality rate (deaths before age 1 per 1000 live births), crude death rate (deaths per 1000 in mean population), crude birth rate (births per 1000 in mean population) in Finland from 1861 to 1873.

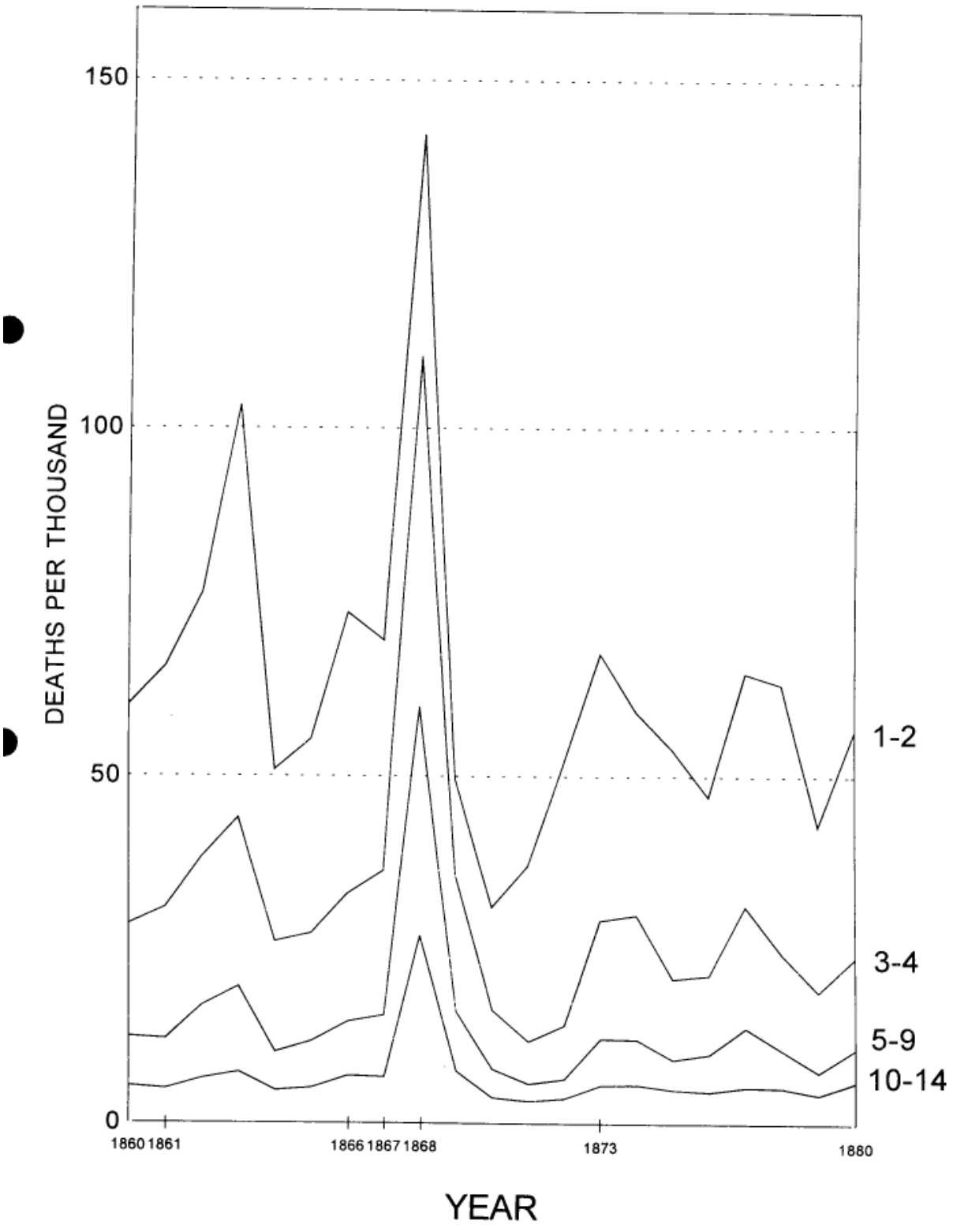
Figure 2. Proportion surviving from birth to age 17 in Finnish cohorts born from 1861 to 1873.

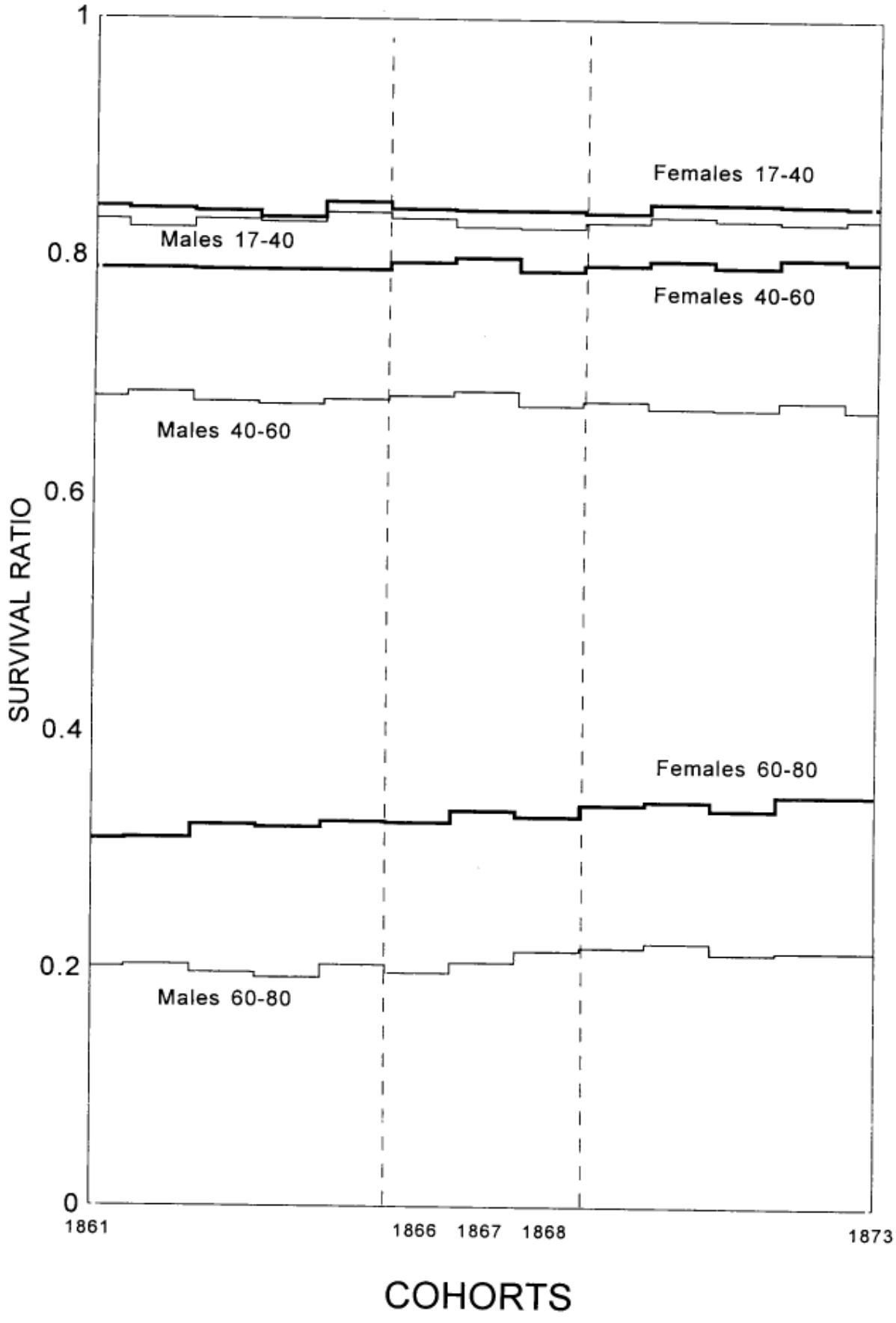
Figure 3. Age-specific death rates (age-groups 1-2, 3-4, 5-9 and 10-14 years) for both sexes in Finland from 1860 to 1880.[11]

Figure 4. Proportion surviving specified age intervals in Finnish cohorts born from 1861 to 1873.


500
400
300
200
100
0
INFANT MORTALITY RATE
100
80
60
40
20
0
CRUDE BIRTH/DEATH RATE
1861
1866
1867
1868
1873
YEAR
CRUDE BIRTH RATE
CRUDE DEATH RATE
INFANT MORTALITY RATE


0.75
0.7
0.65
0.6
0.55
0.5
1861
1866
1867
1868
1873
COHORTS
MALE
FEMALE


150
100
50
0
DEATHS PER THOUSAND
1860 1861
1866 1867 1868
1873
1880
YEAR
1-2
3-4
5-9
10-14


1
0.8
0.6
0.4
0.2
0
SURVIVAL RATIO
Females 17-40
Males 17-40
Females 40-60
Males 40-60
Females 60-80
Males 60-80
1861
1866
1867
1868
1873
COHORTS